

# HÆMODYNAMIC STUDIES DURING AURICULAR FIBRILLATION AND AFTER RESTORATION OF SINUS RHYTHM

BY

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Auricular fibrillation is one of the most frequent rhythm disturbances in organic heart diseases, being present in about 70 per cent of all cases of congestive failure. Sometimes fibrillation is found in young persons with otherwise healthy hearts, as stated already by Mackenzie in 1911; in 651 cases of auricular fibrillation he found no other signs of heart disease in 4.6 per cent. Orgain *et al.* (1936) found this form of fibrillation in 6.5 per cent. Evans and Swann (1954) described this as "lone auricular fibrillation", by which they mean auricular fibrillation without signs of structural heart disease. Conditions for the diagnosis are normal heart size and heart rate and no murmur: the functional capacity and prognosis of the subject are normal.

As a consequence, the regular contractions of the auricles cannot always be essential for the working capacity of the heart. It may, however, represent a less rational way of heart work. In healthy subjects the reserve capacity of the heart is so great that a slight reduction of its efficiency is of minor importance, and will only be noticed under great strain. In the failing heart, however, even a small reduction of the capacity may be of importance. Auricular fibrillation often marks an aggravation of congestive failure in heart disease. However, this is primarily due to the tachycardia or indicates a progression of the heart disease. It is therefore very difficult to separate the effect of the auricular fibrillation *per se* on the symptoms and signs of cardiac patients from the deleterious effect of other factors.

Stewart *et al.* (1928) examined the rate of blood flow during experimental fibrillation in dogs. They found no difference compared to sinus rhythm.

By means of heart catheterization it is possible to study the circulation in man more closely. Ferrer *et al.* (1948) as well as Wade *et al.* (1952) compared two groups of patients with rheumatic heart disease, with and without fibrillation, and found (on the average) lower cardiac output in the cases of fibrillation. It is difficult, however, to draw conclusions from a comparison between two small groups of different patients. Kory and Meneely (1951) studied the circulation by heart catheterization before and after the restoration of normal rhythm with quinidine. In six of eight patients they found a considerable increase of the cardiac output both at rest and during exercise after sinus rhythm was restored. Hecht *et al.* (1951) examined 15 patients in the same way: while at rest the mean increase of the cardiac output after the achievement of sinus rhythm was small, the increase during exercise was statistically significant, and the mean pressure in the pulmonary artery found was unchanged. They concluded that "these observations indicate that a coordinate contraction of the cardiac atria aids in cardiac filling during demand periods, and that this reserve function is lost in atrial fibrillation."

Reeve-Hansen *et al.* (1952) studied 14 patients with fibrillation of durations ranging from six months to five years. All patients had been digitalized. From three weeks to two months after restoration of sinus rhythm by means of quinidine the patients were re-examined. In 9 of 14 patients a significant (i.e. more than 10%) increase of the cardiac output at rest was found, while in five

it was unchanged. The peripheral resistance was significantly reduced in nine. The mean arterial pressure showed some variations. The pressure in the pulmonary artery showed no systematic change. They concluded that the results indicated an improvement of the circulation due to the restoration of sinus rhythm by means of quinidine, and they also maintained that a subjective improvement occurred. Storstein and Tveten (1955) confirmed these findings in five patients. These authors have all found a decline in the arterio-venous oxygen difference after restoration of sinus rhythm. Storstein and Tveten suggested that the effect may be due to the peripheral vasodilating effect of the quinidine with a reduction of the peripheral resistance. According to investigations by Ferrer *et al.* (1948) no unequivocal hæmodynamic changes have been found after quinidine medication to indicate that the drug is the cause of all the changes found after conversion to sinus rhythm independent of the sinus rhythm *per se*.

The findings in these investigations vary so much that further observations are necessary to reach reliable conclusions.

#### MATERIAL AND METHODS

Twenty patients (14 men and 6 women) were examined by heart catheterization before and after restoration of normal rhythm with quinidine. Eleven suffered from mitral stenosis, in five combined with aortic valve disease, four had coronary heart disease, two were of unknown origin, one had thyrotoxicosis, and one lone auricular fibrillation.

The degree of functional capacity was assessed on admittance according to the New York Heart Association's classification. Thirteen belonged to groups I and II, 5 to group III, and 2 to group IV.

All patients had been digitalized for a long time and had spent a sufficiently long period in the ward for the circulation to stabilize. None of them had noticeable congestive failure with œdema before the investigations were started. They had toilet privileges, but were otherwise confined to bed.

Restoration of normal rhythm was carried out with doses of quinidine sulphate increasing from 1.2 g. to as much as 3.6 g. daily. Two suffered relapses, but were later converted to sinus rhythm after a second attempt. The majority of the patients were kept on a maintenance dose of 0.8 g. per day after the conversion, a few somewhat less. In the last seven patients the quinidine medication was stopped for some days before the second catheterization. Otherwise the treatment was the same before and after restoration.

During the heart catheterizations, pressures were recorded in the right atrium, the pulmonary artery and in the pulmonary artery wedge position, and blood samples obtained from a pulmonary and a peripheral artery with the patient resting on the table. The same pressure recordings and blood samples were obtained during exercise on a bicycle ergometer with a fixed resistance, except for observations from the right atrium. The second catheterization was performed after sinus rhythm had lasted from a minimum of four days up to three months, usually after a period of from one to two weeks.

The exercise tests were carried out as uniformly as possible, during auricular fibrillation and sinus rhythm. It was impossible to obtain all the planned observations in all patients. Particularly during the exercise test some blood samples and pressure registrations failed.

The oxygen consumption was determined by means of Douglas bags, and the cardiac output according to the direct Fick principle. For the pressure registrations Tybjerg-Hansen's manometer was employed. The work of the right ventricle against pressure was calculated according to the formula:

$$RV.W = \frac{CO \times 1.055 \times (PAP - RAP) \times 13.6}{1000}$$

where *PAP* is the pressure in the pulmonary artery, *RAP* the pressure in the right atrium and *CO* the cardiac output. Only mean pressures are reported.

## RESULTS

The results are shown in Table I where the mean values during auricular fibrillation are compared with the findings after conversion to sinus rhythm. A statistical analysis of the observations is also given. Only those cases in which comparable values were obtained are included. An evaluation of the work of the right ventricle during exercise was not possible as pressure measurements in the right atrium were carried out only at rest.

TABLE I  
RESULTS OF STATISTICAL ANALYSIS

	n	m		x	S	t	P
Cardiac output (l./min.)		A.F.	S.R.				
Rest .. .. .	17	5.6	6.2	-0.63	0.84	-3.0	0.01 *
Exercise .. ..	11	6.8	8.1	-1.52	0.99	-4.85	0.001 *
Stroke volume (ml.)							
Rest .. .. .	18	66	78	-11.89	19.07	-2.57	0.02 *
Exercise .. ..	9	51	69	-15.78	27.06	-1.65	0.15
O <sub>2</sub> consumption (ml./min.)							
Rest .. .. .	19	257	269	-12.37	37.79	1.39	0.20
Exercise .. ..	13	527	537	10.31	11.08	0.32	0.80
Arterial O <sub>2</sub> saturation (%)							
Rest .. .. .	19	95.9	95.7	0.23	2.06	0.48	0.70
Exercise .. ..	6	94.9	95.1	-0.17	2.43	-0.15	0.90
O <sub>2</sub> saturation, pulmonary artery (%)							
Rest .. .. .	19	71.8	73.6	-1.89	3.06	-2.63	0.02 *
Exercise .. ..	16	57.6	60.9	-3.31	5.58	-2.30	0.03 *
Arterio-venous O <sub>2</sub> difference (vol. %)							
Rest .. .. .	20	4.9	4.15	6.0	8.53	3.09	0.01 *
Exercise .. ..	20	7.4	6.3	10.7	15.92	2.93	0.01 *
Pulmonary artery pressure (mm. Hg)							
Rest .. .. .	19	20.7	21.8	-1.08	4.89	-0.94	0.30
Exercise .. ..	17	30.5	32.8	-2.32	5.76	-1.61	0.10
Pulmonary capillary pressure (mm. Hg)							
Rest .. .. .	16	12.0	14.4	-2.44	3.76	-2.51	0.03 *
Exercise .. ..	12	21.9	25.1	-3.5	6.12	-1.90	0.09
Right ventricular work (kg./min.)							
Rest .. .. .	16	1.35	1.64	-0.29	0.34	-3.3	0.01 *

n=comparable numbers of patients.

m=mean values.

x=mean difference.

- = lower values in auricular fibrillation.

S=variance of the difference.

$t = \text{Student's } t\text{-test} \frac{\bar{x}}{S} \sqrt{n-1}$ .

\* = the difference is of statistical significance.

P=level of significance

S.R.=sinus rhythm.

A.F.=auricular fibrillation.

As might be expected in investigations where the analytical errors are not inconsiderable, variations in both directions were found. However, most of the observations show clear trends in the variations of the haemodynamic conditions after restoration of sinus rhythm, and some definite conclusions may be drawn. The cardiac output increases in the great majority of cases, particularly during exercise. In two cases the increase at rest was great and might be due to analytical errors. They are therefore not included in the statistical analysis.

As might be expected, the increase in cardiac output found was due to an increase of the stroke volume. The oxygen consumption remained practically unchanged. Likewise, there was no

clear trend in the variation of the arterial oxygen saturation, neither at rest nor during exercise. After conversion to sinus rhythm there was in most cases an increase of the oxygen content of blood samples from the pulmonary artery with a corresponding reduction of the arterio-venous oxygen difference both at rest and during exercise. There was no difference in the pressure in the pulmonary artery, but some tendency towards a moderate increase of the pulmonary capillary pressure both at rest and during exercise. The work of the right ventricle clearly increased at rest. Right ventricular work during exercise could not be calculated. However, the greater increase in cardiac output during exercise than at rest, and the constancy of the pulmonary artery pressure indicate a correspondingly greater increase in this work during exercise.

#### DISCUSSION

The increase in cardiac output and stroke volume indicate that sinus rhythm represents a more efficient working of the heart than auricular fibrillation with a greater filling of the ventricles, possibly owing to the auricular activity. The striking increase of the arterio-venous oxygen difference and the decrease in oxygen saturation in mixed venous blood during fibrillation means that, because of a reduced circulation, the peripheral tissues are kept under poorer conditions with regard to oxygen supply and tensions than during sinus rhythm. The improved peripheral conditions during sinus rhythm are at the expense of an increase in the load on the heart, here expressed as a significant increase in work against pressure, and a raised pulmonary venous (capillary) pressure.

To investigate the possible effect of quinidine on the circulation beyond that due to correction of arrhythmia, this drug was withdrawn for some days before the second catheterization in the last seven patients. A statistical analysis showed no difference between this group and the rest of the patients except with regard to the arterio-venous oxygen difference at rest. Those who were given quinidine continuously showed a greater difference after conversion to sinus rhythm ( $P: 0.05$ ). Thus there can be hardly any doubt that the hæmodynamic changes demonstrated are due to the restoration of sinus rhythm.

Of the patients examined about half suffered from mitral stenosis. These on the whole had a greater reduction of functional capacity than the others. The patients with mitral stenosis have been compared with the rest of the material in order to see whether they reacted differently to the restoration. Only two observations showed a difference of statistical significance between the two groups. The group with mitral stenosis, compared with the rest, showed after restoration of sinus rhythm: (1) a greater increase in stroke volume at rest ( $P: 0.001$ ) and during exercise ( $P: 0.05$ ), and (2) less increase in the oxygen saturation in the pulmonary artery at rest ( $P: 0.05$ ). It accordingly does not appear as if a transition from auricular fibrillation to sinus rhythm gives changes of greater importance for the circulatory dynamics in cases of mitral stenosis than in fibrillation from other causes.

The investigations show with certainty that a change in the hæmodynamic conditions takes place after resumption of sinus rhythm. That this is favourable for the peripheral circulation cannot be doubted, even if some patients may show a fairly large increase of the pulmonary capillary pressure, particularly during exercise.

To a heart with great functional capacity these changes are certainly of no particular importance. In a subject with an otherwise healthy heart abolition of an existing auricular fibrillation may hardly cause a subjectively noticeable improvement of his functional capacity. To the failing heart, however, the clearly demonstrated changes in the working capacity of the heart may be of great importance.

As far as possible our therapy should therefore aim at achieving sinus rhythm. However, one must be aware that the pulmonary capillary pressure, particularly during exercise, may rise seriously. In three patients the mean pressure during exercise increased by 10.5, 11, and 12 mm. mercury and reached levels where the danger of pulmonary œdema is considerable (Gorlin *et al.*, 1951).

## SUMMARY AND CONCLUSIONS

Twenty patients with auricular fibrillation due to various causes have, after preliminary treatment with digitalis, been catheterized and the circulation studied. After restoration of normal rhythm with quinidine, catheterization has been repeated. The oxygen consumption was essentially unchanged, likewise the arterial oxygen saturation. The other results showed that cardiac output and stroke volume increased both at rest and during exercise after restoration of sinus rhythm. Simultaneously, the oxygen content of the pulmonary artery blood increased, and the arterio-venous oxygen difference decreased considerably. The pressure in the pulmonary artery remained unchanged, but the pulmonary capillary pressure rose after conversion to sinus rhythm both at rest and during exercise, the increase during exercise being not statistically significant. A reduction of the pulmonary vascular resistance was therefore highly probable. The work performed by the right ventricle at rest increased noticeably.

In seven patients the quinidine was withheld for some days before the last catheterization. These patients reacted in about the same way as the others. The only difference of statistical significance was in the arterio-venous oxygen difference at rest. Those who were given quinidine continuously showed a greater difference after regaining sinus rhythm. The quinidine is accordingly not the cause of the hæmodynamic changes observed after transition from auricular fibrillation to sinus rhythm has occurred.

Eleven patients with mitral stenosis were compared with the remaining nine patients. The changes observed were the same, except that in mitral stenosis the stroke volume showed a greater increase at rest and during exercise and the oxygen saturation of mixed venous blood less increase at rest after conversion to sinus rhythm.

Our conclusions are that the circulation improves after the restoration of sinus rhythm, but that the pulmonary capillary pressure in some cases increases to an unfavourable degree.

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